Cardiac Excitability and Cardiac Conduction

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ILOs:

By the end of this lecture the student should be able to:

- Describe cardiac excitability ARP and RRP
- Correlate the myocardial action potential with the myocardial excitability and contraction
- Explain the effects of altered serum calcium and potassium levels on the myocardial excitability
- Analyze with illustration the propagated cardiac impulse along the conductive system of the heart and its component
- Explain the causes and significances of slow conduction velocity at the AVN

Excitability

- It is ability of myocardial cells to generate action potentials in response to inward, depolarizing current.

Refractoriness

It is the inability to respond to a second stimulus if it arrives during the action potential.

The refractory periods reflect differences in excitability over the duration of the action potential

Excitability changes in the fast response cardiac fibers

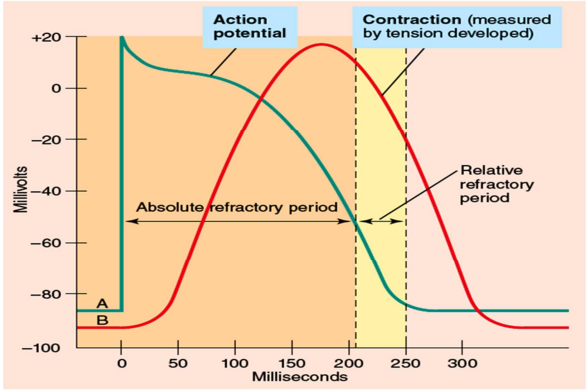
Absolute Refractory Period (ARP):

- ARP is the time during which the cardiac cells are inexcitable and cannot respond even to stronger stimuli and a second propagated action potential cannot be generated.
- The ARP extends from beginning of phase 0 to the middle of phase 3.

- It is due to inactivation of the fast voltage-gated Na^+ channels, so no Na^+ current.
- It is also called effective refractory period (ERP).
- It coincides with the systole and beginning of diastole.
- Significance of ARP:
    - The cardiac muscle has a long refractory period (250 to 300 msec.) because of the prolonged plateau phase of the action potential.
    - So, cardiac muscle cannot be restimulated until contraction is almost over, preventing summation of contractions and tetanus of cardiac muscle.
    - Thus, long ARP protects the heart against the danger of tetanization by allowing more relaxation and more filling before the ventricle can respond to the second impulse (complete tetanization prevents the heart filling which only occurs during relaxation).

Relative Refractory Period (RRP)

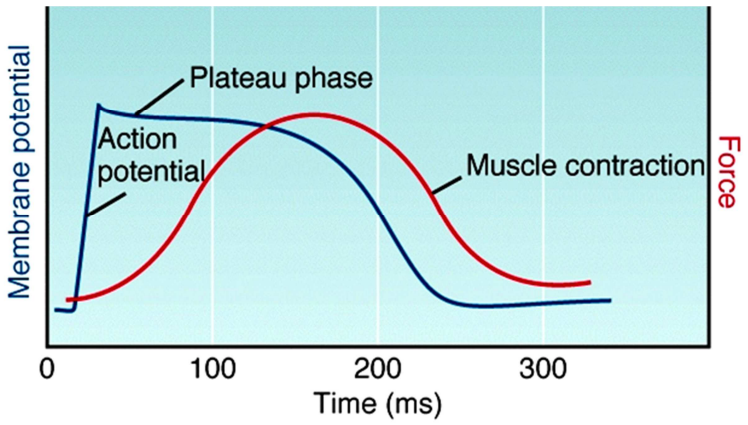
- There is gradual recovery of excitability but is still below normal.
- Cells can respond only to stimuli that are a stronger than normal.
- RRP extends from middle of phase 3 till its end (end of repolarization phase).
- During this RRP, an AP can be elicited by a stronger stimulus.
- It is caused by reactivation of some of the fast Na^+ channels.
- As the membrane potential repolarizes, an increasing number of Na^+ channels become activated.
- It coincides with the first half of diastole.



Excitability changes during fast response action potential

Relationship between cardiac electric (AP) and mechanical response

- Contraction (systole) begins just after the start of depolarization.
- Systole reaches its maximum at last 1/3 of the plateau.
- First half of diastole coincides with phase 3 (rapid repolarization).
- Second half of diastole coincides with phase 4.



Relationship between cardiac electric (AP) and mechanical response

Effects of altered serum potassium and calcium levels on the myocardial excitability

I. Changes in blood potassium levels

Hypokalemia

- Decrease K^+ in ECF
- → ↑ K^+ concentration gradient across cell membrane
- → Stimulates passive flow of K^+
- → More K^+ goes out the cell
- → more negative RMP
- → ↓ cell excitability due to its hyperpolarization.

Hyperkalemia

- Increased extracellular K^+ ions (hyperkalemia) is a very dangerous and potentially lethal condition.
- The effect of hyperkalemia on cardiac excitability is complex
- While initial increase in K^+ → ↑ excitability (by an opposite mechanism to hypokalemia)

- Further increase in K^+ has an opposite effect
- → ↓ cell excitability and may stop heart in diastole because
  - ↓ K^+ efflux from myocardial cell
  - → less negative RMP & cardiac cells cannot repolarize.
  - This means that cardiac cells are depolarized.
  - → This inactivates Na^+ channels
  - → slower influx of Na^+ and cells cannot fire
  - → ↓ amplitude of action potential
  - → ↓ influx of Ca^{2+}
  - → heart cannot contract

II. Changes in blood calcium levels

Hypercalcemia

- Increased extracellular Ca^{2+} (Hypercalcemia) decreases excitability because it blocks sodium movement through voltage –gated Na^+ channels. This leads to decrease in Na+ entry into the cells delaying the threshold potential and decrease generation of action potential.

Hypocalcemia

- Cardiac excitability increases by opposite mechanism.
- Decreased extracellular Ca^{2+} facilitates Na^+ entry into the cells, shifting the membrane potential to more positive value, (reaching the threshold potential faster) and the cells become more excitable.

Conductivity

- It is the ability of cardiac muscle to transmit action potential from one cell of heart to next.

The heart contracts or beats rhythmically as a result of action potential that originates in a specialized **part of cardiac conduction system** (pacemaker = SAN) and spreads via this system to all parts of the myocardium.

The structures that make up the conduction system are the **sinoatrial node (SA node),** the **internodal atrial pathways,** the **atrioventricular node (AV node),** the **bundle of His** and its branches, and the **Purkinje system.**

- All the cardiac muscle cells are conductive but at different rates.
  - ► In the Purkinje system, the rate is fast (4 m /sec)
  - ► In atrial pathways, ventricular muscle and bundle of His, rate is 1 m /sec.
  - ► In the nodal tissue, the rate of conduction is very slow (0.05 m /sec).

The velocity of conduction of impulses is determined by size of fibers and gap junctions

- The velocity of conduction is highest in Purkinje system because it has large fiber size and numerous gap junctions (very high level of permeability so ions are transmitted easily from one cell to the next).
- The velocity of conduction is slowest in nodal tissue (especially the AVN) because it has small fiber size and few gap junctions (great resistance to conduction of excitatory ions from one conducting fiber to the next).

Conduction of Impulses in Cardiac Muscle

- Normally, the action potential of the heart is initiated in the SAN.
- Action potentials that originate in the SAN spread to the right atrium then to the left atrium and AVN. The wave passes through 3 tracts in atria called internodal atrial tracts (the anterior, middle and posterior tracts). Also, conduction occurs through atrial myocytes, but is slower than in these tracts.
- Because the atria and ventricles are separated by the fibrous skeleton of the heart which is electrically nonconductive, the impulse cannot be conducted directly from the atria to the ventricles. AVN is the only pathway for spread of action potential between atria and ventricles.
- The conduction of impulse in AVN is delayed about 0.1sec between atria and ventricles. This is called AV nodal delay. This delay is to allow atria to contract and empty their content of blood in the ventricle before the ventricle depolarizes and contract. This allows time for complete ventricular filling before contraction.

- Action potentials continue through the atrioventricular bundle, or bundle of His, that starts at the AVN and enters the interventricular septum.
- Bundle of His then divides into right and left bundle branches that transmit the impulse rapidly down the septum.
- The right and left bundle branches run down either side of the septum to apex of the heart then reflected upwards along the lateral walls of ventricles to the base of the heart.
- Each bundle ends by small fibers called Purkinje fibers that spread to all parts of the ventricular myocardium (ventricular muscle). Within the myocardium of the ventricles, the action potential spreads from the inner (endocardium) to the outer (epicardium) side. The action potential, also, spreads from one ventricular muscle cell to the next.
- Purkinje fibers have the maximum conduction velocity (4 m/sec). This rapid conduction of the action potential throughout the ventricles causes simultaneous activation of both ventricles and allows for efficient contraction and ejection of blood into both the systemic and pulmonary circulations at the same time.

Important N.B.

- ► First part of the ventricle to be excited is the mid portion of the interventricular septum from left to right.
- ► The last parts of the heart to be depolarized are the posterobasal portion of the left ventricle, the pulmonary conus, and the uppermost portion of the interventricular septum.
- ► Sympathetic stimulation shortens AV nodal delay and speeds conduction whereas parasympathetic stimulation lengthens AV nodal delay and decrease rate of conduction.
- ► Refractory periods are shortened by sympathetic stimulation and prolonged by parasympathetic stimulation.